Rhesus DLL4 / Delta-like 4 Protein (His Tag)

Catalog Number: **90882-C08H**

Sino Biological
Biological Solution Specialist

General Information

Gene Name Synonym:

DLL4

Protein Construction:

A DNA sequence encoding the rhesus DLL4 (XP_001099250.1) (Met1-Pro528) was expressed with a polyhistidine tag at the C-terminus.

Source: Rhesus

Expression Host: HEK293 Cells

QC Testing

Purity: > 95 % as determined by SDS-PAGE.

Bio Activity:

Measured by the ability of the immobilized protein to enhance BMP2-induced alkaline phosphatase activity in C3H10T1/2 mouse embryonic fibroblast cells. The ED_{50} for this effect is typically 1-5 μg/mL in the presence of 500 ng/mL recombinant human BMP2.

Endotoxin:

< 1.0 EU per μg protein as determined by the LAL method.

Stability:

Samples are stable for up to twelve months from date of receipt at -70 ℃

Predicted N terminal: Ser 27

Molecular Mass:

The recombinant rhesus DLL4 consists of 513 amino acids and predicts a molecular mass of 56.1 kDa.

Formulation:

Lyophilized from sterile 20 mM Tris, 150 mM NaCl, 5 % Glycerol, pH 8.0.

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:

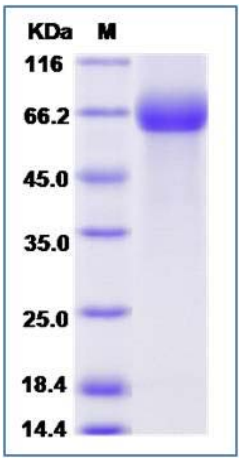
Store it under sterile conditions at -20℃ to -80℃ upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

Delta-like protein 4 (DLL4, Delta4), a type I membrane-bound Notch ligand, is one of five known Notch ligands in mammals and interacts predominantly with Notch 1, which has a key role in vascular development. Recent studies yield substantial insights into the role of DLL4 in angiogenesis. DLL4 is induced by vascular endothelial growth factor (VEGF) and acts downstream of VEGF as a 'brake' on VEGF-induced vessel growth, forming an autoregulatory negative feedback loop inactivating VEGF. DLL4 is downstream of VEGF signaling and its activation triggers a negative feedback that restrains the effects of VEGF. Attenuation of DLL4/Notch signaling results in chaotic vascular network with excessive branching and sprouting. DLL4 is widely distributed in tissues other than vessels including many malignancies. Furthermore, the molecule is internalized on binding its receptor and often transported to the nucleus. In pathological conditions, such as cancer, DLL4 is up-regulated strongly in the tumour vasculature. Blockade of DLL4-mediated Notch signaling strikingly increases nonproductive angiogenesis, but significantly inhibits tumor growth in preclinical mouse models. In preclinical studies, blocking of DLL4/Notch signaling is associated with a paradoxical increase in tumor vessel density, yet causes marked growth inhibition due to functionally defective vasculature. Thus, DLL4 blockade holds promise as an additional strategy for angiogenesis-based cancer therapy.

References

1.Yan M, *et al.* (2007) Delta-like 4/Notch signaling and its therapeutic implications. Clin Cancer Res. 13(24): 7243-6. 2.Sainson RC, *et al.* (2007) Anti-Dll4 therapy: can we block tumour growth by increasing angiogenesis? Trends Mol Med. 13(9): 389-95. 3.Martinez JC, *et al.* (2009) Nuclear and membrane expression of the angiogenesis regulator delta-like ligand 4 (DLL4) in normal and malignant human tissues. Histopathology. 54(5): 598-606.